Mitochondria Functions and Training

Mitochondria Functions — More mitochondria mean more PBs, but what do you have to do to get them?

Deep inside your muscles lurk a multitude of microscopic structures called mitochondria. Although infinitesimally small (they can't be seen with an ordinary microscope), the mitochondria are of major importance to your athletic efforts; as you increase their density, your performance capacity rises concomitantly.

That's because the mitochondria are the only places inside your muscle cells where carbohydrate, fat, and protein can be broken down in the presence of oxygen to create the energy you need to exercise. To put it simply, the more mitochondria you have, the more energy you can generate during exercise, and the faster and longer you can run, cycle or swim.

Intense scientific interest into the function of mitochondria during exercise dates back to the early 1950s, when physiologists noticed that the breast and wing muscles of chickens had few mitochondria, while those of pigeons and mallards contained high densities of the little structures. Of course, chickens can't fly, while mallards and pigeons are the endurance athletes of the bird world, leading researchers to believe that mitochondrial concentrations were closely related to exercise capacity.

Scientists were somewhat surprised to learn that mitochondria contain their own genetic material - and that all the mitochondria in an individual's body are inherited from one's mother, not father (this is because the egg contains mitochondria, while sperm cells are mitochondria-free). This may seem strange, since the egg is rather immobile and the sperm are distance swimmers, but the bottom line is that sperm are so tiny that mitochondria would weigh them down excessively on their harrowing passage toward the egg. The consequence of this, of course, is that you tend to inherit your exercise capacity from your mother, not your dad. If mum is a great endurance athlete, you tend to be one, too, while if dad is a sluggard, it doesn't matter too much.

Of course, scientists began fooling around with ways to increase mitochondrial densities. At first, it was believed that the mitochondria might be under hormonal control, and early research efforts were indeed able to show that mitochondrial numbers were increased when levels of a key hormone produced by the thyroid gland - thyroxine - increased. In laboratory rats, the simple addition of desiccated thyroid to normal rat food caused an explosive increase in mitochondrial size and density in both the heart and liver. Interest in thyroxine as a potential ergogenic aid increased temporarily, until it was discovered that above-normal concentrations of the hormone could produce some very undesirable side effects.

Training and multiplication

It was left to venerated exercise physiologist John Holloszy of the Washington University School of Medicine in St. Louis to show that chronic exercise could put mitochondrial numbers on the upswing. Holloszy simply asked one group of lab rats to run on treadmills for up to 120 minutes per day at intensities of about 50 to 75 per cent of VO2max for a period of 12 weeks, while a second group lolled in their cages. At the end of the 12-week period, Holloszy found that the running rats had increased their mitochondrial densities by approximately 50 to 60 per cent and had also doubled their concentrations of 'cyto-

chrome c,' a key compound found inside mitochondria which is crucially important in aerobic energy production ('Effects of Exercise on Mitochondrial Oxygen Uptake and Respiratory Enzyme Activity in Skeletal Muscle,' The Journal of Biological Chemistry, vol. 242(9), pp. 2278-2282, 1967).

Of course, exercise physiologists then began wondering which type of training was best for perking up mitochondrial numbers. Should one train fast? Long and slow? Mix fast efforts with slow ones? How long should one exercise (how many miles per workout and week) in order to optimise mitochondrial density?

Holloszy and his co-workers at Washington University were the first to really tackle this question. In a fairly simple piece of experimental work, Holloszy et al had one group of rats running 10 minutes per day, another running for 30 minutes, a third group exercising for 60 minutes, and a fourth working for 120 minutes per day. Training took place five days a week for 13 weeks, and training intensity was fixed at about 1.2 mph (or about 32 metres per minute and 313 minutes for the 10K, which is an intensity of around 50- to 60-per cent VO2max for a healthy lab rat).

Not too surprisingly, the two-hour per day runners turned out to have the best mitochondrial setups. For example, compared to sedentary rats, the 10-minute per day exercisers had about 16-per cent more cytochrome c, while the 30-minute workers boosted cytochrome c by 31 per cent. However, rats who ran for an hour expanded cytochrome c by 38 per cent, and the two-hour rats increased it by 92 per cent!

Holloszy's study provided nice support for the specificity of training principle, too, for during a rugged endurance test staged at the end of the research period, the 10-minute rats lasted 22 minutes, the 30-minute ones for 41 minutes, the hour-long rats ran strenuously for 50 minutes, and the two-hour trainees stayed on the treadmills for a whopping 111 minutes! Of course, run time to exhaustion was directly related to cytochrome c concentration; the more c a rat had, the longer it could run at a tough pace ('Skeletal Muscle Respiratory Capacity, Endurance, and Glycogen Utilization,' American Journal of Physiology, vol. 228(4), pp. 1029-1033, 1975).

But What about intensity?

Holloszy's research was great, but it was also limited in application. The key problem, of course, was that he and his colleagues did not look at intensity of training as a mitochondrial-promoting factor, since all of his rats ran at the same speed. However, this research was used by many coaches and experts to prop up the idea that long-duration training (up to two hours per workout or more) was the best way to expand mitochondrial numbers and thereby enhance performance capacity. The philosophy of long, slow distance was the inevitable outcome of this research, and to this day coaches and running gurus sermonise about the critical importance of high-volume, moderate-intensity training for producing optimal 'aerobic adaptations' (meaning, essentially, more mitochondria and thus a higher aerobic capacity) in muscle cells. This philosophy is even carried to the point of absurdity by some exercise scientists, who claim that too high an intensity of training may actually destroy mitochondria.

So, it was up to other researchers to explore the intensity question, and Gary Dudley and his colleagues at the State University of New York at Syracuse did just that. Like Holloszy, Dudley had his rats training five times a week and used a variety of different workout durations, from five minutes up to 90 minutes per day. However, unlike Holloszy, Dudley restricted his study to only eight weeks and used a range of different training intensities — 100% VO2max, 85% VO2max, 70% VO2max, 50% VO2max, and 40% VO2max. Dudley also looked at how different intensities and durations influenced different muscle fibre types (fast twitch, aerobic fast twitch or 'intermediate', and slow twitch), which no one had ever done before ('Influence of Exercise Intensity and Duration on Biochemical Adaptations in Skeletal Muscle,' Journal of Applied Physiology, vol. 53(4), pp. 844-850, 1982).

In contrast to what Holloszy had found, Dudley was able to show that training beyond about 60 minutes per workout was without benefit in terms of increasing cytochrome c. In other words, a

rat training at about 70 to 75% VO2max could upgrade cytochrome c by expanding workout duration from 30 to 60 minutes - but not by increasing workouts from 60 to 90 minutes. This was true at all intensities studied by Dudley - and also with all three muscle fibre types. Progressing beyond about 60 minutes per workout simply didn't have much value when it came to the mitochondria.

The faster you train, the better

However, Dudley's most interesting findings were those related to intensity of training. The Syracuse researcher was able to show that in fast-twitch muscle fibres, just 10 minutes of fast running (at close to 100% VO2max) per day was enough to roughly triple cytochrome c concentrations over an eight-week period. In contrast, running for 27 minutes at 85% VO2max daily only hoisted cytochrome c by 80 per cent, while 60 to 90 minutes at 70 to 75% VO2max nudged cytochrome c upward by just 74 per cent. So much for the theory that intense exercise can hurt mitochondria.

In intermediate muscle cells (those which are roughly half-way between fast twitch and slow twitch), a similar potency of intensity was detected. For example, just 10 minutes of fast running per day fattened cytochrome c as much as 27 minutes daily at 85% VO2max or 60 to 90 minutes at 70 to 75% VO2max. One can only think that a slightly greater amount of fast running would have given speed a definite mitochondrial edge over longer-duration exertions.

When it came to the slow twitch cells, however, the results were a bit different. As mentioned, running more than 60 minutes per workout had no positive effect at all on cytochrome c expansion. The best strategy for slow-twitch, cytochrome-c uplifting turned out to be running about 60 minutes per workout at 70 to 75% VO2max (or around 80 to 84 per cent of max heart rate), which hoisted cytochrome c by approximately 40 per cent. Gamboling along for 27 minutes at 85% VO2max was not far behind, producing a 28-per cent upturn. Fast running at close to 100% VO2max lifted slow twitch cytochrome c by around 10 per cent, a comparatively small gain but one that is not too surprising, given the fact that slow twitch fibres tend to be relied on less heavily than fast twitch cells during fast running. Not to belabour the point, but a 10-per cent increase is not consistent with the idea that fast training is 'hard' on the mitochondria in slow twitch muscles.

And, let's face it, a 10-per cent gain in the slow twitch fibres for 10 minutes of fast running represents an improvement of about 1 per cent per minute. In comparison, running at 85% VO2max lifted cytochrome c in slow-twitch fibres by the same 1-per cent per minute rate, and chugging along at 70 to 75% improved the mitochondria by just 40/60 or 2/3 of a per cent per minute. Again, one has to think that larger amounts of fast running would have pushed the mitochondrial gains toward those observed with slower running.

So what's the bottom line? As Dudley and his colleagues put it, an increase in the intensity of training brings about the greatest adaptive response in the mitochondria. Expressing the crucial importance of intensity another way, Dudley and co-workers said, 'For the same adaptive response, the length of daily exercise necessary to bring about the change becomes less as the intensity of exercise is increased.' In other words, 10 to 15 minutes of running at 5-K pace in a workout can do much more for you than running for 60 to 90 minutes at slower intensities.

Realistically, of course, it's difficult to train fast every day, so almost every athlete ends up with a balance of training, with some days hard and some easy. It's nice to know, however, that to significantly upgrade your muscle-cells' mitochondria, and therefore your VO2max and average racing and training speeds, you don't have to spend hour after hour trudging along. Upswings in your training speed are generally more productive than big upturns in total mileage. Gradual increases in your training intensity — even adding just a few minutes of faster working each day — can pay off with big muscle adaptations and excellent new PBs for you!

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